REMARKS

The application has been amended. In particular, claim 57 has been amended to ensure the proper understanding of the term "cells". Claim 64 has been amended to change the lower end of a concentration range for anisomycin from 0.76 μM to 0.755 μM. Support for the anisomycin concentration range recited in claim 64 can be found in FIG. 16 (A), for example. Also, new claims 65 and 66 are presented herewith. Support for the new claims can be found in the application, for example, in FIG. 15 (A and B). Entry of these amendments and reconsideration are respectfully requested.

Restriction Requirement

In the Advisory Action mailed March 28, 2005, the Examiner has indicated that Applicants' Amendment and Response (filed March 4, 2005) would likely result in a rejoinder of Groups I-II as set forth in the Office Action mailed October 15, 2004. However, the amendment was not entered since the Examiner alleges that claim 64 raises the issue of new matter and requires further consideration and a new search. Applicants respectfully request entry of the amendments filed March 4, 2005 for the reasons set forth below.

Applicants have amended claim 64 in an effort to remove even the suggestion of new matter. Support for the anisomycin concentration range recited in claim 64 can be found, for example, in FIG. 16 (A) of the application. Applicants do not believe that the limitation presented in claim 64 necessitates a new search. In particular, the Examiner has already completely searched and co-examined Applicants' original claims 1-7. Therefore, the Examiner is likely to also have performed a search to claim 64, and it would seem that any relevant documents would have already been found and considered.

In view of the amendments filed March 4, 2005, and the amendments and remarks presented herewith, Applicants respectfully request the rejoinder of Groups I-II as set forth in the Office Action of October 15, 2004.

Claim Rejections Under 35 U.S.C. §112, First and Second Paragraphs

In the Advisory Action dated March 28, 2005, the Examiner has indicated that the claims presented in Applicants' Amendment and Response (filed March 4, 2005) would appear to overcome the 35 U.S.C. §112, first and second paragraph rejections as set forth in the Office Action mailed October 15, 2004. However, as mentioned above, the amendments were not entered since the Examiner is of the opinion that Applicants' claim 64 raises an issue of new matter and requires further consideration and a new search. The Examiner also states in the Advisory Action that a new rejection of claim 57 under 35 U.S.C §112, second paragraph would be necessitated by entry of the amendments of March 4, 2005, since it is allegedly not clear whether the eukaryotic cells are infected with a virus or not.

As described above, support for the language in claim 64 can be found in the application, for example, in FIG. 16 (A). Therefore, contrary to the Examiner's statements, claim 64 does not introduce new matter. Also, as described above, by performing a complete search and co-examination of original claims 1-7, the Examiner is likely to have already performed a search directed to claim 64. Thus, it would seem that any relevant documents would have already been found and considered.

With respect to the new rejection of claim 57, this claim has now been amended to ensure the proper understanding of the term "cells". These amendments are non-narrowing, and simply clarify for the Examiner that the cells are infected with a virus using -1 ribosomal frameshifting. Support for these amendments can be found in the application, for example at page 11, lines 13-26, page 64 lines 7-13 and Figure 15, where #69 HIV gpt human host cells, which are infected with HIV, are treated with anisomycin or sparsomycin.

In view of the amendments to the claims filed March 4, 2005, and the amendments and arguments presented herewith, withdrawal of the 35 U.S.C. 112, first and second paragraph rejections is respectfully requested.

Claim Rejections Under 35 U.S.C.§102 and 35 U.S.C.§102/103

In the Advisory Action mailed March 28, 2005, the Examiner states that in view of the non-entry of the amendment (filed March 4, 2005), applicants arguments in that amendment are not found persuasive to overcome the outstanding rejections as set forth in the Office Action mailed October 15, 2004 for the reasons of record stated therein. The Examiner also states that "even if the amendment were entered, the rejection under 35 U.S.C.§102(b) and the rejection under 35 U.S.C.§102(b)/103 would likely be maintained".

By way of review, the present application was filed with 31 claims. The original claim listing is shown in Exhibit A In the Office Action mailed May 14, 2004, the Examiner agreed to co-examine original claims 1-7. The Examiner rejected claims 1-5 and 7 under 35 U.S.C.§102(b) as allegedly being anticipated by JP 63146818A, as evidenced by Dinman, et al. His stated reasons of record at item [11] of the May 14, 2004 Office Action were as follows:

> **JP 63146818A teaches a method for treating viral infections by administering anisomycin. Dinman, et al. teach anisomycin is a peptidly transferase inhibitor. This anticipates claims 1-5 and 7 as written.**
>
> **While the reference of Dinman, et al. does not antedate the filing date of the application, this reference demonstrates that the ability of anisomycin to affect a eukaryotic peptidyl transferase center is an inherent property of anisomycin. Therefore, the method of treating viral infection by administering anisomycin as taught by JP 63146818 would inherently have affected a eukaryotic peptidyl transferase center.**

Moreover, in the Office Action mailed May 14, 2004, the Examiner rejected claim 6 under 35 U.S.C.§102(b) as allegedly being anticipated by or, in the alternative, under 35 U.S.C.§103(a) as allegedly being obvious over JP 63146818A as evidenced by Dinman, et al. His stated reasons of record at item [12] of the May 14, 2004 Office Action were as follows:

> **Claim 6 is drawn to a method for treating HIV infection by modulating the function of a eukaryotic peptidyl transferase center by administering a drug that affects a eukaryotic peptidyl transferase center. Japanese Patent JP 63146818 further teaches that an example of a "virus" that anisomycin is useful for treating is AIDS (see translated abstract). At the time fo the invention, one would have recognized that AIDS is not a viral infection, but is the manifestation of HIV infection. Thus, one of ordinary skill would have recognized that anisomycin would have inhibited HIV viral replication and not AIDS.**

In applicants' Amendment and Response filed August 11, 2004, the claims were amended. In particular, claims 1 and 7 were amended, and claims 32-56 were added, as shown in Exhibit B.

In the final Office Action mailed October 15, 2004, the Examiner maintained the rejection of claims 1-5 and also rejected claims 32, 34, 44-46, 51 and 53 under 35 U.S.C. 102(b) as being allegedly anticipated by JP 63146818 as evidenced by Dinman for the reasons of record as set forth at item [11] of the Office Action mailed May 14, 2004, and for the reasons set forth at item [27] of the final Office Action mailed October 15, 204.

Under item [27] in the final Office Action, the Examiner responds to applicants' argument in the Response filed August 11, 2005 that JP 63146818 A fails to teach or suggest

administering a drug so as to specifically affect ribosomal frameshifting and/or nonsense-mediated mRNA decay. In particular, the Examiner states that:

> **... there is no requirement in claim 1 that the administered drug actually affect programmed ribosomal frameshifting and/or nonsense-mediated mRNA decay.**

He also states at item [27] in the final Office Action that:

> **Even assuming *arguendo* the claim was so limited to the drug affecting programmed ribosomal frameshifting and/or nonsense-mediated mRNA decay, it is noted that this is an inherent feature of practicing the method of treating HIV by administering anisomycin as taught by JP 63146818 A.**

Moreover, in the final Office Action, the Examiner responds to Applicants' argument in the Response filed August 11, 2004 that Dinman, et al. was published after the earliest effective filing date of the instant application and cannot be used against the present claims. In particular, the Examiner refers to MPEP 2131.01 and MPEP 2124 and states that:

> **... the reference of Dinman, et al., teaching a universal fact, i.e., that anisomycin is a peptidyl transferase inhibitor, need not antedate the effective filing date of the instant application**

Also, in the final Office Action, the Examiner maintained the rejection of claim 6 and also rejected claim 47 under 35 U.S.C. §102(b) as allegedly being anticipated by or, in the alternative, under 35 U.S.C. §103(a) as allegedly being obvious over JP 63146818 A, as evidenced by Dinman, et al. for reasons of record as set forth at item [12] of the Office Action mailed May 14, 2004, and for the reasons stated in the final Office Action (at item [27]).

In Applicants' Amendment and Response filed March 4, 2005, Applicants attempted to replace the listing of claims with the listing shown in Exhibit C. However, in the Advisory Action, the Examiner did not enter these amendments, alleging that new claim 64 raises an issue of new matter, and requires further consideration and a new search. Also, in the Advisory Action, the Examiner states that he would likely maintain the 35 U.S.C.§102/§102/103 rejections.

As described above, support for the language in claim 64 can be found in the application, for example, in FIG. 16 (A). Therefore, contrary to the Examiner's statements, claim 64 does not introduce new matter. Also, as described above, by performing a complete search and co-examination of original claims 1-7, the Examiner is likely to have already performed a search directed to claim 64. Thus, it would seem that any relevant documents would have already been found and considered. In view of these facts, Applicants have requested entry of the amendments filed on March 4, 2005, and have also requested entry of the amendments presented herewith.

That said, the 35 U.S.C §102 and 35 U.S.C.§102/103 claim rejections will be addressed together with respect to currently amended independent claim 57, which reads as follows:

57. A method comprising treating eukaryotic infections caused by viruses using programmed -1 ribosomal frameshifting by exposing eukaryotic cells infected with a virus using said -1 ribosomal frameshifting to a compound selected from anisomycin and sparsomycin, wherein the compound modulates the efficiency of programmed -1 ribosomal frameshifting, thereby suppressing viral propagation in the cells.

Whereas the Examiner has stated that he would likely maintain the 35 U.S.C.§102/§102/103 rejections, no reasons other than reasons of record are provided. It is respectfully submitted that the Examiner's reasons of record fail to sufficiently address each of the elements of claim 57.

For example, as outlined above, the Examiner previously stated that "JP 63146818A teaches a method for treating viral infections by administering anisomycin", and that "Japanese Patent JP 63146818 further teaches that an example of a "virus" that anisomycin is useful for treating is AIDS".

However, there is nothing in these alleged teachings which expressly discloses or suggests treating eukaryotic infections caused by viruses using programmed -1 ribosomal frameshifting by the use of anisomycin or any other compound. In fact, there is no mention of -1 ribosomal frameshifting in the JP patent. Since JP 63146818 A fails to disclose this element of claim 57, Applicants submit that maintaining the §102/§102/103 rejections would be improper.

Applicants agree that the JP patent provides a general teaching to use anisomycin to treat viral infections. However, a general teaching to use anisomycin to treat infections caused by a laundry list of DNA and RNA viruses, many of which do not use programmed -1 ribosomal frameshifting, actually teaches away from the present invention. Applicants have found that anisomycin and sparsomycin cannot be used to treat just any virus, and that it will not work on some of the viruses included in the families listed in the JP patent. For example, it is not effective against retroviruses using +1 ribosomal frameshifting.

Whereas Applicants agree that the JP patent lists AIDS among the viruses that can be treated with anisomycin, it is noted that it also lists virtually all DNA and RNA virus families known in the art. Such a laundry list disclosure would not have reasonably led the skilled

artisan to viruses using programmed -1 ribosomal frameshifting. As the Examiner is aware, he must consider the teachings of the JP patent as a whole.

Also, even if affecting programmed -1 ribosomal frameshifting "is an inherent feature of practicing the method of treating HIV by administering anisomycin as taught by JP 63146818 A", as the Examiner has alleged (see outline above), this is not a proper basis for rejecting the claimed invention. In particular, that which is inherent in the prior art, if not known at the time of the invention, cannot form a proper basis for the rejection. Before the present invention, nothing in the art suggested employing anisomycin or sparsomycin against viruses using the -1 ribosomal frameshifting mechanism as opposed to just any virus.

Furthermore, the JP patent is not an enabled reference for use against the invention as presently claimed. For example, the teachings of the JP patent are so broad as to encompass treating virtually any viral infection, including those caused by viruses that don't use -1 ribosomal frameshifting.

Also, there is a complete lack in the JP patent of any working examples directed to the treatment of infections causes by viruses employing the -1 ribosomal frameshifting mechanism. Without having performed the kinds of extensive *in vivo* and *in vitro* studies that Applicants have performed, it would not be reasonable for a skilled artisan to say that anisomycin or sparsomycin would affect the efficiency of programmed -1 ribosomal frameshifting and thus be effective against viruses using this mechanism.

In fact, a review of the English portions of the JP patent indicates that they provide only two examples (see page 143). These examples are directed to coxsackie virus and herpes simplex virus, which don't employ -1 ribosomal frameshifting. Thus, that which may be enabled (treatment of coxsackie and herpes simplex infections), would have led the skilled artisan away from the invention as claimed.

Based on the content of the disclosure of the JP patent, the quantity of experimentation that would have been needed before a skilled artisan would use anisomycin (or sparsomycin) to treat viral infections causes by viruses using -1 ribosomal frameshifting would have been undue. Also, the skilled artisan would recognize that the ability to successfully treat a particular type of viral infection would depend upon the compound. Therefore, it would have been highly unpredictable as to whether the guidance and working examples for treatment of coxsackie and herpes simplex viral infections would translate into a successful method of treatment for infections causes by viruses employing the -1 ribosomal frameshifting mechanism.

Finally, in moving from the prior art to the claimed invention, the proper test for patentability requires determining what the prior art would have led the skilled person to do, and not what they might try or find obvious to try. Applicants submit that the JP patent would not have led the skilled person to use anisomycin (or sparsomycin) to treat viruses using -1 ribosomal frameshifting given the lack of enablement of the JP patent with respect to such teachings. In fact, it would have led the skilled person in a direction which is not claimed, since the working examples are directed to viruses that don't use this mechanism.

The Examiner has relied upon the Dinman, et al. reference for evidence showing that anisomycin is a peptidly transferase inhibitor. The Examiner alleges that Dinman, et al. can be used to show evidence of a "universal fact", even though it was published more than 1 1/2 years after Applicants' earliest priority date and is based, in part, on the present invention. Regardless, before the present invention, the public was not in possession of the method presently recited in claim 57. Therefore, no further discussion is believed necessary with regard to the Dinman, et al. reference.

The Examiner's attention is drawn to new claims 65 and 66. The present inventors have found that anisomycin and sparsomycin are particularly effective at treating infections caused by viruses using -1 ribosomal frameshifting over those caused by viruses that don't employ this

mechanism. In this regard, Applicants note that in Figure 15A, anisomycin/sparsomycin concentrations of only 1 ng/ml are effective to reduce HIV titers in human cells infected with HIV by about 70-80%, whilst the tables in the JP patent appear to show that at least 0.125 μg/ml (i.e., 100 times more) anisomycin is required to have a similar effect on herpes simplex or coxsackie virus levels.

In view of Applicants' amendments and arguments, Applicants respectfully request withdrawal of these rejections.

Summary

Applicants submit that the claims, as presently recited, are patentably distinct over the art and allowable in form. An allowance of the claims is respectfully requested. Should the Examiner have any questions concerning this Response, he is encouraged to contact the undersigned agent at the telephone number set forth below.

The Commissioner is hereby authorized to charge payment of any additional fees associated with this communication, or credit any overpayment, to Deposit Account No. 08-2461.

Respectfully submitted,

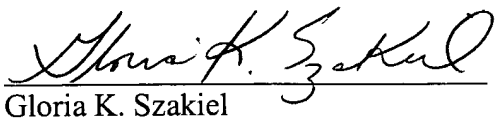

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EXHIBIT A

1368-10 DIV
LISTING OF ORIGINAL CLAIMS

LISTING OF CLAIMS:

1. A method for modulating function of a eukaryotic peptidyl transferase center comprising administering a drug that affects the eukaryotic peptidyl transferase center.

2. The method according to claim 1, wherein the drug is an antibiotic.

3. The method according to claim 1, wherein the drug is a peptidyl transferase center inhibitor.

4. The method according to claim 2, wherein the drug is selected from the group consisting of sparsomycin and anisomycin.

5. A method for treating a viral infection comprising modulating the function of a eukayotic peptidyl transferase center according to the method of claim 1.

6. A method for treating HIV infection comprising modulating the function of a eukaryotic peptidyl transferase center according to the method of claim 4.

7. A method for treating a disease associated with a nonsense mutation in a gene modulating the function of a eukaryotic peptidyl transferase center according to the method of claim 1.

8. A mutant gene encoding a protein involved in ribosomal frameshifting, wherein the mutation results in modulation of the efficiency of ribosomal frameshifting.

9. The mutant gene of claim 8, which increases the efficiency of ribosomal frameshifting.

10. The mutant gene of claim 8, which modulates nonsense mRNA decay.

11. The mutant gene of claim 8, selected from the group consisting of *mof4-1*, *mof2-1*, and *mof5-1*.

12. An expression vector comprising a mutant gene of claim 8 operatively associated with an expression control sequence.

13. A method for modulating ribosomal frameshifting or mRNA decay comprising introducing an expression vector of claim 12 into cells.

14. A method for treating a viral infection comprising modulating ribosomal frameshifting according to the method of claim 13.

15. A method for treating a disease associated with a nonsense mutation in a gene comprising modulating mRNA decay according to the method of claim 13.

16. A nucleic acid hybridizable *in vivo* with a mRNA coding for a protein involved in programmed -1 ribosomal frameshifting, wherein a mutation of a gene encoding the protein results in modulation of the efficiency of ribosomal frameshifting.

17. The nucleic acid of claim 16, which is selected from the group consisting of an antisense nucleic acid and a ribozyme.

18. The nucleic acid of claim 17, wherein the gene is selected from the group consisting of *mof4-1*, *mof2-1*, and *mof5-1*.

19. An expression vector comprising a nucleic acid of claim 17 operatively associated with an expression control sequence.

20. A method for modulating ribosomal frameshifting or mRNA decay comprising introducing an expression vector of claim 19 into cells.

21. A method for treating a viral infection comprising modulating ribosomal frameshifting according to the method of claim 20.

22. A method for treating a disease associated with a nonsense mutation in a gene comprising modulating mRNA decay according to the method of claim 20.

23. A method of screening for a drug active in the eukaryotic peptidyl transferase center comprising:

a) contacting cells with a candidate drug; and

b) assaying for modulation of peptidyl transferases;

wherein a drug that modulates peptidyl transferases is active in the eukaryotic peptidyl transferase center.

24. The method according to claim 23, wherein the modulation of peptidyl transferases is assayed by a method selected from the group consisting of:

i) identifying a phenotype associated with a mutation selected from the group consisting of *mof1-1*, *mof4-1*, *mof2-1*, *mof5-1*, *mof6-1*, and *his4*;

ii) detecting increasing stability of nonsense mRNA or short mRNA transcripts;

iii) culturing a mutant cell line deficient in tyrosine and leucine biosynthesis on a tyrosine and leucine deficient culture medium;

iv) detecting an altered ratio of Gag to Gag-pol proteins in a virus infected cell; and

v) binding to a protein that modulates a frameshifting event.

25. A polypeptide corresponding to the N-terminal 100 amino acids of ribosomal binding protein L3.

26. A method for increasing the efficiency of -1, but not of +1 ribosomal frameshifting, comprising introducing the polypeptide of claim 25 into a cell.

27. A method for treating a viral infection comprising modulating ribosomal frameshifting according to the method of claim 26.

28. A nucleic acid encoding the polypeptide of claim 25.

29. An expression vector comprising a nucleic acid of claim 28 operatively associated with an expression control sequence.

30. A method for modulating ribosomal frameshifting comprising introducing an expression vector of claim 29 into cells.

31. A method for treating a viral infection comprising modulating ribosomal frameshifting according to the method of claim 30.

EXHIBIT B

1368-10 DIV
LISTING OF CLAIMS IN THE
AMENDMENT AND RESPONSE OF AUGUST 11, 2004

LISTING OF CLAIMS:

1. (currently amended) A method for treating disorders associated with the activities of ~~modulating function of~~ a eukaryotic peptidyl transferase center comprising administering ~~a drug that affects the eukaryotic peptidyl transferase center~~ to a patient in need thereof a therapeutically effective amount of a drug which modulates programmed ribosomal frameshifting and/or nonsense-mediated mRNA decay.

2. (original) The method according to claim 1, wherein the drug is an antibiotic.

3. (original) The method according to claim 1, wherein the drug is a peptidyl transferase center inhibitor.

4. (original) The method according to claim 2, wherein the drug is selected from the group consisting of sparsomycin and anisomycin.

5. (original) A method for treating a viral infection comprising modulating a function of a eukayotic peptidyl transferase center according to the method of claim 1.

6. (original) A method for treating HIV infection comprising modulating the function of a eukaryotic peptidyl transferase center according to the method of claim 1.

7. (currently amended) A method for treating a disease ~~associated with~~ resulting from a nonsense mutation in a gene comprising modulating the function of a eukaryotic peptidyl transferase center according to the method of claim 1.

8-31. (cancelled)

32. (new) The method according to claim 1, wherein the drug modulates the efficiency of -1 ribosomal frameshifting.

33. (new) The method according to claim 32, wherein the drug increases the efficiency of -1 ribosomal frameshifting.

34. (new) The method according to claim 32, wherein the drug decreases the efficiency of -1 ribosomal frameshifting.

35. (new) The method according to claim 1, wherein the drug suppresses a nonsense mutation.

36. (new) The method according to claim 1, wherein the drug stabilizes a nonsense transcript.

37. (new) The method according to claim 1, wherein the drug interacts with a protein encoded by a gene selected from the group consisting of *mof4-1*, *mof2-1*, *mof5-1* and human homologues thereof.

38. (new) The method according to claim 1, wherein the drug is polypeptide of a ribosome binding protein, L3.

39. (new) The method according to claim 1, wherein the drug is a vector comprising a gene encoding a protein involved in programmed ribosomal frameshifting and/or nonsense-mediated mRNA decay.

40. (new) The method according to claim 39, wherein the gene is selected from the group consisting of *mof4-1*, *mof2-1*, *mof5-1* and human homologues thereof.

41. (new) The method according to claim 39, wherein the vector is a viral or retroviral vector.

42. (new) The method according to claim 1, wherein the drug is an expression vector comprising a nucleic acid hybridizable *in vivo* with an mRNA encoding a protein involved in programmed ribosomal frameshifting and/or nonsense-mediated mRNA decay, wherein a mutation of a gene encoding the protein changes the efficiency of ribosomal frameshifting.

43. (new) The method according to claim 42, wherein the hybridizable nucleic acid is an antisense RNA specific for the mRNA, the antisense RNA being operatively associated with an expression control sequence.

44. (new) The method according to claim 5, wherein the drug inhibits viral propagation.

45. (new) The method according to claim 5, wherein the drug affects programmed ribosomal frameshifting in an RNA virus.

46. (new) The method according to claim 45, wherein the RNA virus is selected from the group consisting of retroviruses, astroviruses and totiviruses.

47. (new) The method according to claim 6, wherein the drug inhibits viral propagation.

48. (new) The method of claim 7, wherein the drug suppresses a nonsense mutation.

49. (new) The method of claim 7, wherein the drug stabilizes a nonsense transcript.

50. (new) The method of claim 7, wherein the disease is selected from the group consisting of nonspherocytic hemolytic anemia, β-thalassemia, hypercholesterolemia, pulmonary emphysema, adrenal hyperplasia, apolipoprotein C-II deficiency, hemophilia B, Bernard-Soulier syndrome, fructose intolerance, insulin resistance, maple syrup urine disease, thrombosis, goiter and hypothyroidism, chronic gradulomatous, Sandhoff disease, vonWillebrand disease type III, gyrate atrophy, 1,25-dihydroxyvitamine D3 resistant rickets, spherocytosis, cystic fibrosis and spherocytosis.

51. (new) A method for inhibiting the function of a eukaryotic peptidyl transferase center, the method comprising exposing cells to an effective amount of drug, under conditions for a sufficient time to change the efficiency of -1 ribosomal frameshifting and/or suppress a nonsense mutation.

52. (new) The method of claim 51, wherein the drug increases the efficiency of -1 ribosomal frameshifting.

53. (new) The method of claim 51, wherein the cells are infected with an RNA virus and the drug inhibits propagation of the RNA virus.

54. (new) The method of claim 51, wherein the cells contain a gene carrying the nonsense mutation, which results in a disease.

55. (new) The method of claim 54, wherein the disease is selected from the group consisting of nonspherocytic hemolytic anemia, β-thalassemia, hypercholesterolemia, pulmonary emphysema, adrenal hyperplasia, apolipoprotein C-II deficiency, hemophilia B, Bernard-Soulier syndrome, fructose intolerance, insulin resistance, maple syrup urine disease, thrombosis, goiter and hypothyroidism, chronic gradulomatous, Sandhoff disease, vonWillebrand disease type III, gyrate atrophy, 1,25-dihydroxyvitamine D3 resistant rickets, spherocytosis, cystic fibrosis and spherocytosis.

56. (new) The method of claim 51, wherein the drug stabilizes a nonsense transcript.

EXHIBIT C

1368-10 DIV
LISTING OF CLAIMS IN THE
AMENDMENT AND RESPONSE OF MARCH 4, 2005

LISTING OF CLAIMS:

1-6. (Canceled)

7. (Withdrawn) A method for treating a disease resulting from a nonsense mutation in a gene comprising modulating the function of a eukaryotic peptidyl transferase center according to the method of claim 1.

8-34. (Canceled)

35. (Withdrawn) The method according to claim 1, wherein the drug suppresses a nonsense mutation.

36. (Withdrawn) The method according to claim 1, wherein the drug stabilizes a nonsense transcript.

37. (Withdrawn) The method according to claim 1, wherein the drug interacts with a protein encoded by a gene selected from the group consisting of *mof4-1*, *mof2-1*, *mof5-1* and human homologues thereof.

38. (Withdrawn) The method according to claim 1, wherein the drug is polypeptide of a ribosome binding protein, L3.

39. (Withdrawn) The method according to claim 1, wherein the drug is a vector comprising a gene encoding a protein involved in programmed ribosomal frameshifting and/or nonsense-mediated mRNA decay.

40. (Withdrawn) The method according to claim 39, wherein the gene is selected from the group consisting of *mof4-1*, *mof2-1*, *mof5-1* and human homologues thereof.

41. (Withdrawn) The method according to claim 39, wherein the vector is a viral or retroviral vector.

42. (Withdrawn) The method according to claim 1, wherein the drug is an expression vector comprising a nucleic acid hybridizable *in vivo* with an mRNA encoding a protein involved in programmed ribosomal frameshifting and/or nonsense-mediated mRNA decay, wherein a mutation of a gene encoding the protein changes the efficiency of ribosomal frameshifting.

43. (Withdrawn) The method according to claim 42, wherein the hybridizable nucleic acid is an antisense RNA specific for the mRNA, the antisense RNA being operatively associated with an expression control sequence.

44-47. (Canceled)

48. (Withdrawn) The method of claim 7, wherein the drug suppresses a nonsense mutation.

49. (Withdrawn) The method of claim 7, wherein the drug stabilizes a nonsense transcript.

50. (Withdrawn) The method of claim 7, wherein the disease is selected from the group consisting of nonspherocytic hemolytic anemia, β-thalassemia, hypercholesterolemia, pulmonary emphysema, adrenal hyperplasia, apolipoprotein C-II deficiency, hemophilia B, Bernard-Soulier syndrome, fructose intolerance, insulin resistance, maple syrup urine disease, thrombosis, goiter and hypothyroidism, chronic gradulomatous, Sandhoff disease, vonWillebrand disease type III, gyrate atrophy, 1,25-dihydroxyvitamine D3 resistant rickets, spherocytosis, cystic fibrosis and spherocytosis.

51-53. (Canceled)

54. (Withdrawn) The method of claim 51, wherein the cells contain a gene carrying the nonsense mutation, which results in a disease.

55. (Withdrawn) The method of claim 54, wherein the disease is selected from the group consisting of nonspherocytic hemolytic anemia, β-thalassemia, hypercholesterolemia, pulmonary emphysema, adrenal hyperplasia, apolipoprotein C-II deficiency, hemophilia B, Bernard-Soulier syndrome, fructose intolerance, insulin resistance, maple syrup urine disease, thrombosis, goiter and hypothyroidism, chronic gradulomatous, Sandhoff disease, vonWillebrand disease type III, gyrate atrophy, 1,25-dihydroxyvitamine D3 resistant rickets, spherocytosis, cystic fibrosis and spherocytosis.

56. (Withdrawn) The method of claim 51, wherein the drug stabilizes a nonsense transcript.

57. (New) A method comprising treating eukaryotic infections caused by viruses using programmed -1 ribosomal frameshifting by exposing eukaryotic cells to a compound selected from the group consisting of anisomycin and sparsomycin, wherein the compound modulates the efficiency of programmed -1 ribosomal frameshifting, thereby suppressing viral propagation in the cells.

58. (New) The method of claim 57, wherein the compound modulates programmed -1 ribosomal frameshifting in an RNA virus.

59. (New) The method of claim 57, wherein the viruses are selected from the group consisting of retroviruses, coronaviruses, paramyxoviruses, astroviruses and totiviruses.

60. (New) The method of claim 59, wherein the virus is HIV.

61. (New) The method of claim 57, wherein the compound is sparsomycin, which increases the efficiency of programmed -1 ribosomal frameshifting, thereby suppressing viral propagation.

62. (New) The method of claim 57, wherein the compound is anisomycin, which decreases the efficiency of programmed -1 ribosomal frameshifting, thereby suppressing viral propagation.

63. (New) The method of claim 61, wherein sparsomycin is used in an amount of about 0.52 μM to about 2.6 μM.

64. (New) The method of claim 62, wherein anisomycin is used in an amount of about 0.76 μM to about 3.8 μM.